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☐ 1: Semin Interv Cardiol 2000 Sep;5(3):109-115

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Pathophysiology of coronary thrombosis.

PubMed Services

Kristensen SD, Lassen JF, Ravn HB.

Department of Cardiology B and Institute of Experimental Clinical Research, University Hospital, Skejby Sygehus, Arhus N, Denmark. steendk@dadlnet.dk

Related Resources

Detailed knowledge of the pathophysiology as well as the dynamic nature of thrombus formation provides a valuable tool for correct management and prophylactic adjunctive therapy in patients with acute coronary syndromes. Coronary thrombosis in the majority of cases caused by disruption or fissuring of an atherosclerotic plaque. At the lesion thrombogenic material will be exposed to the flowing blood leading to activation of platelets and the formation of a platelet clot. Simultaneously, the coagulation system is activated resulting in increased thrombin formation. Thrombin is a key mediator in arterial thrombosis, due to its effect on both platelets and fibrin generation. Thrombin contributes to the stabilization of an initially loose platelet aggregate by generating cross-bound fibrin within the thrombus. During the course of an acute coronary syndrome, the patient presents changing chest pain and dynamic ischaemic ECG findings. This is likely to be related to the dynamic nature of the pathophysiology. The presence of a non-occlusive coronary thrombus may decrease the myocardium its normal blood flow and oxygen supply, leading to ischaemia. During lysis or embolization, blood supply may be restored, but the presence of thrombus fragments in the microcirculation holds the potential to sustained interference with myocardial metabolism. The emboli contain activated platelets which release vasoconstrictors that may compromise the microcirculation. Recurrent thrombus formation at the lesion site may result in occlusion of the artery adding to the dynamic nature of the clinical presentation. In conclusion, platelets, the coagulation system, and the endothelium cause a dynamic process of intermittent occlusion, vasospasm and embolization of thrombus material.

Publication Types:

- Review
- Review, tutorial

PMID: 11054908 [PubMed - indexed for MEDLINE]



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Antiplatelet medications and their indications in preventing and treating coronary thrombosis.

Van De Graaff E, Steinhubl SR.

Department of Cardiology, Wilford Hall Medical Center, San Antonio, TX, U

Related Resources

Platelets play a pivotal role in the pathophysiology of unstable angina, acute myocardial infarction, and complications following percutaneous coronary intervention. Three classes of platelet-inhibiting drugs, aspirin, thienopyridine: platelet glycoprotein IIb/ IIIa inhibitors, are now commonly used for the prevention and treatment of disorders of coronary artery thrombosis. For the last several years aspirin has been the sole option for antiplatelet therapy in the treatment and prevention of the manifestations of cardiovascular disease. However, a wider selection of antiplatelet agents, including the thienopyridines (ticlopidine and clopidogrel) and the platelet glycoprotein (GP)IIb/IIIa receptor antagonists, are now available and provide clinicians with the opportunity to potentially improve upon the previous gold standard of aspirin. This review summarizes these drugs and the scientific data that have led to their use in primary and secondary prevention, angina, myocardial infarction, and percutaneous coronary intervention.

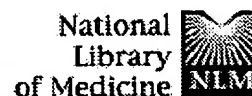
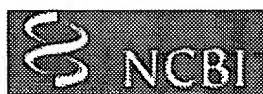
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Management of venous and cardiovascular thrombosis: enoxap

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Harvey DM, Offord RH.

Department of Haematology, Northwick Park Hospital NHS Trust, Harrow, Middlesex.

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Enoxaparin has strong clinical evidence that supports its license in a broad spe of therapeutic indications, including thromboprophylaxis in surgical patients, 1 patients bedridden because of acute illness, the once-daily treatment of venou: thromboembolism and the treatment of unstable angina and non-Q wave myoc infarction.

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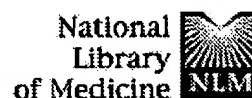
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Nitric oxide insufficiency and arterial thrombosis.

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Loscalzo J, Freedman J, Inbal A, Keaney JF Jr, Michelson AD, Vita JA.

Evans Department of Medicine, Boston University School of Medicine,
Massachusetts 02118-2394, USA.

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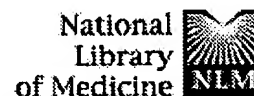
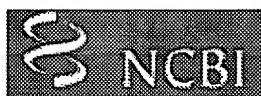
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Direct thrombin inhibitors for treatment of arterial thrombosis potential differences between bivalirudin and hirudin.

Bates SM, Weitz JI.

McMaster University and Hamilton Civic Hospitals Research Centre, Ontario Canada.

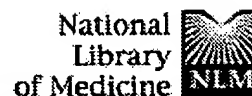
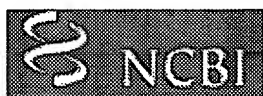
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Given the central role of thrombin in arterial thrombogenesis, most treatment strategies for acute coronary syndromes are aimed at inhibiting its generation blocking its activity. Although heparin has been widely used, it has limitations setting of arterial thrombosis. These limitations reflect the inability of heparin inactivate thrombin bound to fibrin, a major stimulus for thrombus growth. In addition, the anticoagulant response to heparin varies from patient to patient, heparin is neutralized by platelet Factor IV, large quantities of which are relea from platelets activated at sites of plaque rupture. Consequently, heparin requ careful laboratory monitoring to ensure an adequate anticoagulant effect. Dire thrombin inhibitors, such as hirudin and bivalirudin, overcome the limitations heparin. These agents inhibit fibrin-bound thrombin, as well as fluid-phase thr and produce a predictable anticoagulant response. Bivalirudin has both safety potential efficacy advantages over hirudin. Bivalirudin appears to have a wider therapeutic window than hirudin, possibly because bivalirudin only transiently the active site of thrombin. The better safety profile of bivalirudin permits administration of higher doses, which may give it an efficacy advantage. Hiru prevents thrombin from activating protein C, thereby suppressing this natural anticoagulant pathway. In contrast, bivalirudin may promote protein C activat transiently inhibiting thrombin until it can be bound by thrombomodulin. Diffe between bivalirudin and hirudin, as well as other direct thrombin inhibitors, hi the pitfalls of considering all direct thrombin inhibitors to have equivalent risk profiles.

Publication Types:

- Review
- Review, tutorial

PMID: 9809887 [PubMed - indexed for MEDLINE]



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☐ 1: Nephron 1993;63(3):273-278

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Plasma parameters of the prothrombotic state in chronic urem

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Sagripanti A, Cupisti A, Baicchi U, Ferdeghini M, Morelli E, Barsotti G

Istituto Clinica Medica I, Universita di Pisa, Italy.

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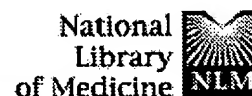
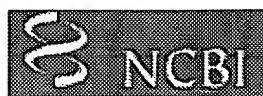
We measured plasma parameters of the prothrombotic state, namely thrombin-antithrombin III complex (TAT), fibrinopeptide A (FPA), D-dimer (von Willebrand factor (vWF), tissue-type plasminogen activator (tPA), beta-thromboglobulin (beta TG), platelet factor 4 (PF4) and serotonin (5HT) series of 51 adult patients with chronic uremia: 22 were on maintenance hemodialysis (MHD) and 29 on conservative dietary treatment. Serum tumor necrosis factor (TNF) was determined as well. Uremics presented significantly higher levels of FPA, DD, vWF, TNF, beta TG and 5HT than normal controls. Patients on conservative treatment showed lower levels of TAT, DD, TNF and beta TG than patients on MHD. Our results provide evidence that a prothrombotic state exists in chronic uremia and that MHD patients have a higher degree of hypercoagulability. Both hemodialysis procedure and uremia-related factors are likely to contribute to the hemostatic derangement.

PMID: 8446263 [PubMed - indexed for MEDLINE]

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☐ 1: Kidney Int 1992 Nov;42(5):1124-1129

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Procoagulant effect of the OKT3 monoclonal antibody: involve of tumor necrosis factor.

Pradier O, Marchant A, Abramowicz D, De Pauw L, Vereerstraeten P, Kinnaert P, Vanherweghem JL, Capel P, Goldman M.

Department of Immunology, Hematology and Transfusion, Hopital Erasme, Universite Libre de Bruxelles, Belgium.

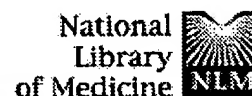
Related Resources

We recently observed that the prophylactic administration of high doses of OKT3 monoclonal antibody (MoAb) in cadaveric renal transplantation favors the development of thromboses of the grafts' main vessels and of thrombotic microangiopathies. These clinical observations led us to perform sequential determinations of plasma levels of prothrombin fragment 1 and 2 (F 1 + 2) and degradation products (FDP) after the first injection of 5 or 10 mg OKT3 given prophylaxis in kidney transplant recipients. The values observed have been compared with those of kidney transplant recipients not treated with OKT3. F 1 + 2 levels peaked four hours after the first injection of 5 mg OKT3 (mean +/- SEM: 4.8 +/- 0.73 vs. 1.75 +/- 0.37 nmol/liter in controls, $P < 0.01$), indicating activation of a common pathway of the coagulation cascade. FDP levels were already above control values at four hours and continued to increase until 24 hours (mean +/- SEM: 4729 +/- 879 vs. 1038 +/- 320 ng/ml in controls, $P < 0.05$), indicating a fibrinolytic process. The magnitude and the time course of the changes in F 1 + 2 and FDP plasma levels were similar whether the patients received 5 or 10 mg dose of OKT3. The levels of von Willebrand factor (VWF) antigen, a molecule released by activated or damaged endothelial cells, were also significantly increased after injection of OKT3 (mean +/- SEM at 24 hr, 3.67 +/- 0.18 vs. 2.17 +/- 0.11 U/ml in controls, $P < 0.05$). The procoagulant effects of OKT3 were further investigated in vitro on human umbilical vein endothelial cells (HUVEC). (ABSTRACT TRUNCATED AT 200 WORDS)

Publication Types:

- Clinical trial
- Controlled clinical trial

PMID: 1453598 [PubMed - indexed for MEDLINE]



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Activation of endothelial cells induces platelet thrombus formation on their matrix. Studies of new in vitro thrombosis model with molecular weight heparin as anticoagulant.

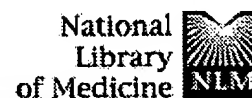
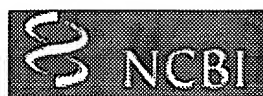
Zwaginga JJ, Sixma JJ, de Groot PG.

Department of Hematology, University Hospital, Utrecht, The Netherlands.

Related Resources

Previous studies have indicated that activation of endothelial cells may lead to production of tissue factor. We have studied the effect of endothelial cell activation and subsequent tissue factor synthesis on thrombus formation on the extracellular matrix in flowing blood. Endothelial cells were stimulated with tumor necrosis factor, endotoxin, or phorbol ester. Coverslips with activated cells or their extracellular matrix were introduced into a perfusion system and exposed to blood anticoagulated with 20 U/ml low molecular weight heparin. This concentration allowed manipulation of blood without activation of the coagulation cascade. Platelet deposition and thrombus formation were evaluated by morphometry, and fibrinopeptide A formation was assayed as a measure of thrombin generation. Activation of endothelial cells caused fibrinopeptide A generation in the perfusate and some deposition of fibrin on the surface of endothelial cells; however, platelets were not deposited. The matrix of the stirred endothelium also caused enhanced fibrinopeptide A generation, and platelet aggregates and fibrin were deposited on the matrix. Maximal effects were observed with stimulation periods between 4 and 10 hours and were still clearly present after 18 hours. Increase in shear rate, perfusion time, and platelet number resulted in an increase in platelet adhesion, but platelet aggregate formation as a percentage of adhesion remained constant. Platelet aggregate formation and fibrinopeptide A generation were inhibited with antibodies against tissue factor or factor VIIa. Platelet aggregate formation alone was inhibited by antibodies against glycoprotein IIIb. Polymerization of fibrin on the matrix was best supported in perfusions at a low shear rate. The new in vitro thrombosis model presented here provides a powerful tool for study of the regulation of thrombogenesis by the vessel wall in response to various stimuli.

PMID: 2297347 [PubMed - indexed for MEDLINE]



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Hormone replacement therapy reduces the reactivity of monocytes and platelets in whole blood--a beneficial effect on atherosclerosis and thrombus formation?

Aune B, Oian P, Omsjo I, Osterud B.

Department of Obstetrics and Gynecology, University of Tromso, Norway.

Related Resources

OBJECTIVE: Our purpose was to investigate the effects of hormone replacement therapy on the reactivity of monocytes and platelets in whole blood, measured tissue factor activity, tumor necrosis factor- α , and thromboxane B2. **STUDY DESIGN:** Thirty-two women were randomized into either transdermal or oral combined hormone replacement therapy and underwent blood sampling before and after 3 and 12 months of treatment. The tissue factor activity in monocytes was measured both in unstimulated whole blood and after a weak lipopolysaccharide stimulation. Tumor necrosis factor- α and thromboxane B2 formation in platelets were measured after a weak lipopolysaccharide stimulation of whole blood. **RESULTS:** After 12 months of hormone replacement therapy there were significant reductions of tissue factor activity in both unstimulated and lipopolysaccharide-stimulated monocytes ($p < 0.001$) and significant reduction in formation of tumor necrosis factor- α ($p < 0.03$) and thromboxane B2 ($p < 0.05$). There were no differences in these parameters between the transdermal and oral groups. No changes were observed after 3 months of therapy. **CONCLUSION:** Twelve months of hormone replacement therapy reduces cellular activation of monocytes and platelets; these changes may account for some of the beneficial effects in reducing the risk of cardiovascular disease.

Publication Types:

- Clinical trial
- Randomized controlled trial

PMID: 8610768 [PubMed - indexed for MEDLINE]

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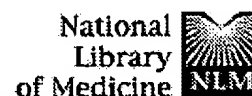
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☐ 1: Am J Cardiol 1991 Sep 3;68(7):36B-50B

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Coronary atherosclerotic plaques with and without thrombus in ischemic heart syndromes: a morphologic, immunohistochemic and biochemical study.

Arbustini E, Grasso M, Diegoli M, Pucci A, Bramerio M, Ardissino D, A L, de Servi S, Bramucci E, Mussini A, et al.

Department of Pathology, Universita di Pavia, Italy.

Related Resources

We investigated incidence, severity, and distribution of coronary atherosclerosis, acute thrombosis, and plaque fissuring in ischemic heart disease (both unstable syndromes and chronic ischemia) and in nonischemic controls. We also studied structural, immunohistochemical, and biochemical profile of plaques, with and without thrombus, including morphometry, immunophenotyping of inflammatory infiltrates, cytokine presence, and ultrastructural features. Critical coronary stenosis was almost the rule in both acute and chronic ischemic series (greater than 90% whereas it reached 50% in control subjects). Thrombosis was principally characteristic of unstable-acute ischemic syndromes (unstable angina, 32%; acute myocardial infarction, 52%; cardiac sudden death, 26%) but was also found in chronic ischemia (stable angina, 12%; ischemic cardiomyopathy, 14%) and in control subjects (10%). Plaque fissuring without thrombus occurred in low percentages in lipid-rich, so-called eccentric plaques in most series. Major differences were found between pultaceous-rich versus fibrous plaques rather than between plaques with or without thrombus. Pultaceous-rich plaques were frequent in sites of critical stenosis, thrombosis, and ulceration. Inflammatory infiltrates, i.e., T cells, macrophages, and few beta cells, mostly occurred in lipid-rich, plaques unrelated to thrombus. In the adventitia, infiltrates were a common finding unrelated to any syndrome. Necrotic cytokines such as alpha-TNF were immunohistochemically detected in macrophages, smooth muscle, and intimal cells and detected by immunoblotting in 67% of pultaceous-rich plaques, either with or without thrombus. Immune response mediators such as IL-2 were also expressed in analogous plaques but in a minor percentage (50%-40%). Media were extensively damaged in severely diseased arteries with and without thrombus. Ultrastructural study showed that the fibrous cap was either highly cellular or densely fibrillar. Intimal injury with collagen exposure was often associated with platelet adhesion, whereas foamy cell exposure was not. In conclusion, investigated parameters were essentially similar in plaques, both v

without thrombus, whereas major differences were found between pultaceous and fibrous plaques. Since platelets adhere to exposed collagen and not to foa the type of exposed substrates could play a major role in thrombosis.

PMID: 1892066 [PubMed - indexed for MEDLINE]

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